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NEWS 2		"Ask CAS" for self-help around the clock
NEWS 3	Jun 03	New e-mail delivery for search results now available
NEWS 4	Aug 08	PHARMAMarketLetter(PHARMAML) - new on STN
NEWS 5	Aug 19	Aquatic Toxicity Information Retrieval (AQUIRE) now available on STN
NEWS 6	Aug 26	Sequence searching in REGISTRY enhanced
NEWS 7	Sep 03	JAPIO has been reloaded and enhanced
NEWS 8	Sep 16	Experimental properties added to the REGISTRY file
NEWS 9	Sep 16	CA Section Thesaurus available in CAPLUS and CA
NEWS 10	Oct 01	CASREACT Enriched with Reactions from 1907 to 1985
NEWS 11	Oct 24	BEILSTEIN adds new search fields
NEWS 12	Oct 24	Nutraceuticals International (NUTRACEUT) now available on STN
NEWS 13	Nov 18	DKILIT has been renamed APOLLIT
NEWS 14	Nov 25	More calculated properties added to REGISTRY
NEWS 15	Dec 04	CSA files on STN
NEWS 16	Dec 17	PCTFULL now covers WP/PCT Applications from 1978 to date
NEWS 17	Dec 17	TOXCENTER enhanced with additional content
NEWS 18	Dec 17	Adis Clinical Trials Insight now available on STN
NEWS 19	Jan 29	Simultaneous left and right truncation added to COMPENDEX, ENERGY, INSPEC
NEWS 20	Feb 13	CANCERLIT is no longer being updated
NEWS 21	Feb 24	METADEX enhancements
NEWS 22	Feb 24	PCTGEN now available on STN
NEWS 23	Feb 24	TEMA now available on STN
NEWS 24	Feb 26	NTIS now allows simultaneous left and right truncation
NEWS 25	Feb 26	PCTFULL now contains images
NEWS 26	Mar 04	SDI PACKAGE for monthly delivery of multifile SDI results
NEWS 27	Mar 20	EVENTLINE will be removed from STN
NEWS 28	Mar 24	PATDPAFULL now available on STN
NEWS 29	Mar 24	Additional information for trade-named substances without structures available in REGISTRY
NEWS 30	Apr 11	Display formats in DGENE enhanced
NEWS 31	Apr 14	MEDLINE Reload
NEWS 32	Apr 17	Polymer searching in REGISTRY enhanced
NEWS 33	Jun 13	Indexing from 1947 to 1956 added to records in CA/CAPLUS
NEWS 34	Apr 21	New current-awareness alert (SDI) frequency in WPIDS/WPINDEX/WPIX
NEWS 35	Apr 28	RDISCLOSURE now available on STN
NEWS 36	May 05	Pharmacokinetic information and systematic chemical names added to PHAR
NEWS 37	May 15	MEDLINE file segment of TOXCENTER reloaded
NEWS 38	May 15	Supporter information for ENCOMPPAT and ENCOMPLIT updated
NEWS 39	May 16	CHEMREACT will be removed from STN
NEWS 40	May 19	Simultaneous left and right truncation added to WSCA
NEWS 41	May 19	RAPRA enhanced with new search field, simultaneous left and right truncation
NEWS 42	Jun 06	Simultaneous left and right truncation added to CBNB

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 NEWS 43  Jun 06  PASCAL enhanced with additional data

 NEWS EXPRESS  April 4 CURRENT WINDOWS VERSION IS V6.01a, CURRENT
               MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
               AND CURRENT DISCOVER FILE IS DATED 01 APRIL 2003
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FILE COVERS 1907 - 13 Jun 2003  VOL 138 ISS 25
FILE LAST UPDATED: 12 Jun 2003  (20030612/ED)

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=> e histamine H4
E1            2     HISTAMINCE/BI
E2        54778     HISTAMINE/BI
E3            0 --> HISTAMINE H4/BI
E4           33     HISTAMINE1/BI
E5           60     HISTAMINE2/BI
E6            1     HISTAMINE2HCL/BI
E7            1     HISTAMINEAND/BI
E8            1     HISTAMINEANTAGONISTIC/BI
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E9            1     HISTAMINEASE/BI
E10           1     HISTAMINEAZO/BI
E11           1     HISTAMINEAZOBENZENE/BI
E12           2     HISTAMINEAZOPROTEIN/BI

=> e histamine4
E13          60     HISTAMINE2/BI
E14           1     HISTAMINE2HCL/BI
E15           0 --> HISTAMINE4/BI
E16           1     HISTAMINEAND/BI
E17           1     HISTAMINEANTAGONISTIC/BI
E18           1     HISTAMINEASE/BI
E19           1     HISTAMINEAZO/BI
E20           1     HISTAMINEAZOBENZENE/BI
E21           2     HISTAMINEAZOPROTEIN/BI
E22           1     HISTAMINEAZOPROTEINS/BI
E23           1     HISTAMINEBEESWAX/BI
E24           4     HISTAMINEBINDING/BI

=> e histamine4/ct
E#   FREQUENCY    AT     TERM
--   ---------    --     ----
E25           0     1     HISTAMINE-SENSITIZING/CT
E26           0     2     HISTAMINE-SENSITIZING FACTOR/CT
E27           0       --> HISTAMINE4/CT
E28           1           HISTAMINELIKE SUBSTANCES/CT
E29           0     1     HISTAMINERGIC/CT
E30           0     2     HISTAMINERGIC AGONISTS/CT
E31           0     2     HISTAMINERGIC ANTAGONISTS/CT
E32           0     2     HISTAMINERGIC H1 RECEPTORS/CT
E33           0     2     HISTAMINERGIC H2 RECEPTORS/CT
E34           0     2     HISTAMINERGIC H3 NEUROTRANSMITTER AGONISTS/CT
E35           0     2     HISTAMINERGIC H3 NEUROTRANSMITTER ANTAGONISTS/CT
E36           0     2     HISTAMINERGIC H3 RECEPTORS/CT

=> e histaminergic h4/ct
E#   FREQUENCY    AT     TERM
--   ---------    --     ----
E37           0     2     HISTAMINERGIC H3 NEUROTRANSMITTER ANTAGONISTS/CT
E38           0     2     HISTAMINERGIC H3 RECEPTORS/CT
E39           0       --> HISTAMINERGIC H4/CT
E40           0     2     HISTAMINERGIC NERVE/CT
E41           0     2     HISTAMINERGIC NERVOUS SYSTEM/CT
E42           0     2     HISTAMINERGIC RECEPTOR-ANTAGONIZING MOL. STRUCTURE-BIO
                          L. ACTIVITY RELATIONSHIP/CT
E43           0     2     HISTAMINERGIC RECEPTORS/CT
E44           0     2     HISTAMINERGIC SYSTEM BRAIN/CT
E45           0     1     HISTAMINEUM/CT
E46           0     1     HISTAMINIC/CT
E47           0     2     HISTAMINIC AGONISTS/CT
E48           0     2     HISTAMINIC H1 AGONISTS/CT

=> e histaminic h4/ct
E#   FREQUENCY    AT     TERM
--   ---------    --     ----
E49           0     2     HISTAMINIC H3 AGONISTS/CT
E50           0     2     HISTAMINIC H3 RECEPTORS/CT
E51           0       --> HISTAMINIC H4/CT
E52           0     2     HISTAMINIC NERVE/CT
E53           0     2     HISTAMINIC NERVOUS SYSTEM/CT
E54           0     2     HISTAMINIC NEUROTRANSMITTER AGONISTS/CT
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E55          0      2      HISTAMINIC RECEPTOR-BLOCKING MOL. STRUCTURE-BIOL. ACTI
                           VITY RELATIONSHIP/CT
E56          0      2      HISTAMINIC RECEPTORS/CT
E57          0      2      HISTAMINIC SYSTEM BRAIN/CT
E58          0      1      HISTAMINUM/CT
E59          1             HISTAMONAS/CT
E60          0      1      HISTAR/CT

=> del sel
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                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     1.25       1.46

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=> s histamine h4
L1           44 HISTAMINE H4

=> s h4
L2        22539 H4

=> s l1 or l2
L3        22539 L1 OR L2

=> s l3 (w) (antagoni? or inhibit? or block?)
L4           38 L3 (W) (ANTAGONI? OR INHIBIT? OR BLOCK?)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5            31 DUP REM L4 (7 DUPLICATES REMOVED)

=> d ibib abs 26-31

L5   ANSWER 26 OF 31  HCAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:         1983:67803  HCAPLUS
DOCUMENT NUMBER:          98:67803
TITLE:                    H4-Isozyme of lactate dehydrogenase in a solution of
                          sodium chloride.  3.  Enzymic activity and pyruvate
                          inhibition
AUTHOR(S):                Yamamoto, Sadaaki
CORPORATE SOURCE:         Radioisot. Cent., Nagoya Univ., Nagoya, Japan
SOURCE:                   International Journal of Biochemistry (1983), 15(2),
                          185-90
                          CODEN: IJBOBV; ISSN: 0020-711X
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   The inhibition of lactate dehydrogenase isoenzyme H4 (I) by pyruvate (II)
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was investigated. Low II concns. (1 mM) inhibited I; the Km for II was higher in 0.5M NaCl than in 0.1M phosphate buffer. Inhibition by II was greater at 20.degree. than at 40.degree., in all buffers. A mechanism for inhibition by II is suggested whereby a quaternary complex (tetrameric I, NADH, and 2 kinds of II) is formed.

L5 ANSWER 27 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1981:599412 HCAPLUS
DOCUMENT NUMBER: 95:199412
TITLE: Modulation by phosphorylation of interaction between calmodulin and histones
AUTHOR(S): Iwasa, Yasushi; Iwasa, Takafumi; Higashi, Kenji; Matsui, Kazuo; Miyamoto, Eishichi
CORPORATE SOURCE: Med. Sch., Kumamoto Univ., Kumamoto, 860, Japan
SOURCE: FEBS Letters (1981), 133(1), 95-8
CODEN: FEBLAL; ISSN: 0014-5793
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The interaction of calmodulin with histones is Ca2+ and charge-d. dependent and is modulated by histone phosphorylation. This was confirmed by studies on the inhibition of Ca2+-sensitive cyclic nucleotide phosphodiesterase (EC 3.1.4.17) (I) by nonphosphorylated and cAMP-dependent protein kinase-phosphorylated histones. That the histone-calmodulin interaction is Ca2+ and charge-d. dependent was indicated by the requirement for both EGTA and NaCl in the elution of histone from a calmodulin-agarose affinity column. Anal. of histone (H1, H2A, H2B, H3, and **H4**) **inhibition** of the Ca2+-supported I activity showed competition between the apoenzyme and histone for calmodulin activity. The influence of histone phosphorylation on this interaction was illustrated by the higher Ki values for I inhibition by phosphohistones, the mode of inhibition being the same as in the case of nonphosphorylated histones. This reaction further supports the role of charge d. in the interaction as phosphate introduction lowers the pos. charge on histones, thereby decreasing the strength of the histone (pos. charged)-calmodulin (neg. charged) interaction, as seen in the change in the Ki for I.

L5 ANSWER 28 OF 31 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 81200272 MEDLINE
DOCUMENT NUMBER: 81200272 PubMed ID: 7232772
TITLE: Peptides isolated from human liver with specific inhibitory effects on reassociation/reactivation of in vitro dissociated lactic dehydrogenase (LDH-M4 and -H4) isozymes.
AUTHOR: Schoenenberger G A; Buser S; Cueni L; Dobeli H; Gillesen D; Lergier W; Schottli G; Tobler H J; Wilson K
SOURCE: REGULATORY PEPTIDES, (1980 Dec) 1 (3) 223-44.
Journal code: 8100479. ISSN: 0167-0115.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198107
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19810720
AB Two different peptides have been purified from human liver, similar to those previously reported (Schoenenberger, G.A., and Wacker, W.E.C. (1966) Biochemistry 5, 1375--1379) to be present in human urine, which may serve as metabolic regulators of lactate dehydrogenase (EC 1.1.1.27) isoenzymes (LDH-M4 = muscle type; LDH-H4 = heart type). By trichloroacetic acid precipitation, ultrafiltration, Sephadex G-25 and Bio-Gel P-2 columns,

affinity chromatography on immobilized LDH-isozymes and HPLC two peptides which differed with respect to molecular weight, retention on the affinity columns and amino acid composition were isolated. No effect was observed when native, tetrameric lactate dehydrogenase was incubated with these peptides. However, when lactate dehydrogenase was dissociated to monomers at low pH and allowed to reassociate by adjusting the pH to 7.5 complete inhibition of the reactivation occurred when the inhibitors were incubated together with respective reassociating monomeric isozymes. The two peptides showed no cross-specificity, i.e. each peptide exhibited inhibitory activity only on one of the two isozymes LDH-M4 or LDH-H4. From the amino acid analyses, gel filtrations and PAGE + SDS, molecular weights of 1800 for the M4 and approximately 2700 for the **H4 inhibitor** were calculated. An apparent Ki of approximately 3 X 10(-5) mM for the H4 and approximately 7 X 10(-5) mM for the **H4 inhibitor** was estimated. The interaction of the inhibitors with the enzyme system showed strong cooperativity with Hill coefficients of 2.9 (LDH-M4-specific) and 2.4 (LDH-H4-specific). Mathematical modelling of the reassociation and reactivation of lactate dehydrogenase and its specific inhibition by the peptides led to the conclusion that the peptides react with monomers, dimers or a transition state during the tetramerisation process. kappa 1 for the dimerisation step of M4 = 2.0 X 10(5) M-1 . S-1 and of H4 = 8.2 X 10(4) M-1 . S-1; kappa 2 for the tetramerisation step of M4 = 2.8 X 10(5) M-1 . S-1 and of H4 = 1.2 X 10(5) . M-1 S-1, were calculated, the second step still being the faster one (Rudolf, R. and Jaenicke, R. (1976) Eur. J Biochem. 63, 409--417).

L5 ANSWER 29 OF 31 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 80182610 MEDLINE
DOCUMENT NUMBER: 80182610 PubMed ID: 397223
TITLE: Measure of immunoglobulin G-, M-, and A-specific titers against Legionella pneumophila and inhibition of titers against nonspecific, gram-negative bacterial antigens in the indirect immunofluorescence test for legionellosis.
AUTHOR: Wilkinson H W; Farshy C E; Fikes B J; Cruce D D; Yealy L P
SOURCE: JOURNAL OF CLINICAL MICROBIOLOGY, (1979 Nov) 10 (5) 685-9. Journal code: 7505564. ISSN: 0095-1137.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198007
ENTRY DATE: Entered STN: 19900315
Last Updated on STN: 19900315
Entered Medline: 19800728

AB A crude extract of Escherichia coli O13:K92:**H4 inhibited** 97% of positive indirect immunofluorescence titers against a variety of gram-negative bacterial antigens while lowering Legionella pneumophila titers in only 6% of sera from patients with suspected legionellosis. Legionella-specific titers were the result of immunoglobulins G, M, and A, singly or in combination.

L5 ANSWER 30 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1976:161063 HCAPLUS
DOCUMENT NUMBER: 84:161063
TITLE: A study on the properties of H4-LDH partially purified from the cardiac muscle of rabbits
AUTHOR(S): Park, Soo-Hoon; Kimm, Seung-Won
CORPORATE SOURCE: Coll. Med., Seoul Natl. Univ., Seoul, S. Korea
SOURCE: Soul Uidae Chapchi (1975), 16(1), 33-43
CODEN: SUICAC; ISSN: 0582-6802
DOCUMENT TYPE: Journal

LANGUAGE: Korean

AB Lactate dehydrogenase isoenzyme H4 (I) was partially purified .apprx.12-fold from the cardiac muscle of rabbits by means of salting-out with (NH4)2SO4 and of DEAE-cellulose column chromatog. I was inhibited markedly by 0.02M pyruvate to 11% of its original activity. S2-, when added to the reaction system using lactate as the substrate, reduced slightly the inhibition caused by pyruvate. Cysteine, on the other hand, lowered significantly the magnitude of pyruvate inhibition in its lower range of concn., but showed its own inhibitory effect regardless of the pyruvate inhibition in its higher range of concn. The activity of I using lactate as the substrate was more inhibited by the addn. of S2- than by the addn. of 9 4-fold excess of cysteine. When pyruvate was used as substrate, cysteine showed an apparently allosteric effect, while S2- showed no particular effect different from the pyruvate induction itself.

L5 ANSWER 31 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1975:120802 HCAPLUS
DOCUMENT NUMBER: 82:120802
TITLE: Quality control of NADH. Evaluation of methods for detection of inhibitors and specifications for NADH quality
AUTHOR(S): Gerhardt, W.; Kofoed, B.; Westlund, L.; Pavlu, B.
CORPORATE SOURCE: Rigshosp., Univ. Copenhagen, Copenhagen, Den.
SOURCE: Scandinavian Journal of Clinical and Laboratory Investigation, Supplement (1974), 33(139), 51 pp.
CODEN: SCLSAH; ISSN: 0085-591X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Formation of dehydrogenase inhibitors and other contaminants in solid samples of NADH exposed to humidity under controlled conditions was studied, and the stability of NADH stored in several buffers at 37.degree., 25.degree., 4.degree., and -20.degree. was investigated. Increasing concn. of humidity-induced inhibitor was found to correlate pos. to: increasing 260 nm/340 nm absorbance ratio, increasing relative residual 340 nm absorbance increasing relative residual fluorescence, increasing inhibition of lactate dehydrogenase isoenzyme H4 (I) reaction rate with pyruvate, and decreasing pyruvate/2-oxobutyrate I reaction rate ratio. At 50% relative humidity, the following gases increased the rate of NADH destruction: CO2 > air > O2 > N2. Kinetically, the humidity-induced inhibitor was competitive with NADH. On the basis of the linear relationship found between relative residual fluorescence and 260 nm/340 nm absorbance ratio, a predicted 260 nm/340 nm absorbance ratio of 2.24-2.28 was calcd. for a pure NADH prepn. Tris buffer rather than phosphate buffers should be use as solvents for NADH. The following specifications for a ref. NADH are suggested: relative residual absorbance at 340 nm <0.01; relative residual fluorescence <0.01; 260 nm/340 nm absorbance ratio .ltoreq.2.30; pyruvate/2-oxobutyrate reactor rate ratio .gtoreq.1.40.

=> d ibib abs 20-25

L5 ANSWER 20 OF 31 MEDLINE
ACCESSION NUMBER: 88195838 MEDLINE
DOCUMENT NUMBER: 88195838 PubMed ID: 3359929
TITLE: [Histone H4--an opiate antagonist].
Giston **H4--antagonist** opiatov.
AUTHOR: Bagrov A Ia
SOURCE: DOKLADY AKADEMII NAUK SSSR, (1988 Jan-Feb) 298 (1) 240-2.
Journal code: 7505465. ISSN: 0002-3264.
PUB. COUNTRY: USSR

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Russian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198806
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880608

L5 ANSWER 21 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1987:492547 HCAPLUS
DOCUMENT NUMBER: 107:92547
TITLE: Least-squares fitting of tabular data to rational functions in BASIC
AUTHOR(S): Papamichael, Emmanuel M.
CORPORATE SOURCE: Dep. Chem., Univ. Ioannina, Ioannina, 451 10, Greece
SOURCE: Analyst (Cambridge, United Kingdom) (1987), 112(6), 815-19
CODEN: ANALAO; ISSN: 0003-2654
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A theor. explanation of the discontinuous kinetic behavior of polymeric enzymes during inhibition of their reactivity was presented previously. Such a discontinuity was obsd. for the lactate dehydrogenase isoenzyme H4 when its reactivity was inhibited by oxalic and formic acids (conversion of pyruvate to lactate). A simple BASIC program was used to fit the exptl. kinetic data of these reactions with an approximating function by the method of least squares. The program was suitable for the routine simulation of exptl. data with theor. functions.

L5 ANSWER 22 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1987:29951 HCAPLUS
DOCUMENT NUMBER: 106:29951
TITLE: Effect of halocin H4 on cells of Halobacterium halobium
AUTHOR(S): Meseguer, Inmaculada; Rodriguez-Valera, F.
CORPORATE SOURCE: Fac. Med., Univ. Alicante, Alicante, Spain
SOURCE: Journal of General Microbiology (1986), 132(11), 3061-8
CODEN: JGMIAN; ISSN: 0022-1287
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The killing of a population of a sensitive strain of H. halobium by halocin H4 followed exponential kinetics, and the percentage survival of sensitive cells exposed to different concns. of halocin H4 corresponded to single-hit-type kinetics. Morphol. changes were obsd. in treated cells, which showed swollen, spherical shapes. Halocin H4 affected macromol. synthesis very little, and only late after the start of the treatment, although the transport of 2-aminoisobutyric acid, a nonmetabolizable amino acid, was rapidly stopped. Bacteriorhodopsin-mediated H+ extrusion worked very efficiently in treated cells, and much larger pH decreases were found in treated than in untreated suspensions after illumination, although ATP synthesis was not markedly affected. The primary target of halocin H4 may be located in the membrane, producing permeability changes and ionic imbalance, which lead to death and cell lysis.

L5 ANSWER 23 OF 31 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 83160996 MEDLINE
DOCUMENT NUMBER: 83160996 PubMed ID: 6300081
TITLE: Activation of a cyclic AMP-independent protein kinase by an endogenous ATP-requiring protease from lymphosarcoma cells.
AUTHOR: de la Hóussaye B A; Echols T K; Masaracchia R A

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1983 Apr 10) 258 (7) 4272-8.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198305
ENTRY DATE: Entered STN: 19900318
Last Updated on STN: 20021008
Entered Medline: 19830505

AB The activation of a cyclic AMP-independent protein kinase by an endogenous protease is described. The H4 phosphotransferase (Masaracchia, R. A., Kemp, B., and Walsh, D. A. (1977) J. Biol. Chem. 252, 7109-7117) from lymphosarcoma cells was isolated in a nonactive form. Activation required ATP and Mg2+ and was shown to be time-dependent. Although Mn2+ was capable of substituting for Mg2+ in the protein kinase reaction, no activation was observed when Mn2+ replaced Mg2+. The protein substrate histone **H4 inhibited** phosphotransferase activation at concentrations greater than 60 microM. The inhibition was complete in the presence of 100 microM H4. Comparable concentrations of bovine serum albumin did not inhibit the activation. The selective dependence on Mg2+ suggested separate activating and phosphotransferase activities. This was confirmed by heat denaturation in which the activation reaction was shown to be more sensitive to heat inactivation than was the phosphotransferase reaction. The activating enzyme was separated from the protein kinase by column chromatofocusing in the pH range 7-4. The pI of the activating enzyme was greater than 7.0. The pI values of the activated and nonactivated phosphotransferase were 4.8 and 5.3, respectively. The apparent molecular weight of the nonactivated phosphotransferase was 68,000; the activated enzyme was eluted from an S-200 Sephadex column with an apparent Mr = 52,000. Despite many similarities to a protease-activated Ca2+/phospholipid-dependent enzyme isolated from lymphocytes (Ogawa, Y., Takai, Y., Kawahara, Y., Kimura, S., and Nishizuka, Y. (1981) J. Immunol. 127, 1369-1374), the H4 phosphotransferase was not activated by Ca2+, phospholipids, or any combination thereof.

L5 ANSWER 24 OF 31 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 84023838 MEDLINE
DOCUMENT NUMBER: 84023838 PubMed ID: 6226289
TITLE: Histones H3 and **H4 inhibit** protein kinase C specifically.
AUTHOR: Sahyoun N; LeVine H 3rd; Bronson D; McConnell R; Cuatrecasas P
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1983 Sep 30) 115 (3) 1027-32.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198311
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19831123

AB The lysine-rich histone H1 is a preferred substrate for the Ca2+-phospholipid-dependent protein kinase (protein kinase C). Histones H3 and H4 are poor substrates but potent inhibitors of the enzyme. The inhibitory effect of H3 and H4 seems to result mainly from a decreased sensitivity of protein kinase C to stimulation by phosphatidylserine (PS).

These observations suggest that site-specific phosphorylation of one histone type can be regulated by other histones.

L5 ANSWER 25 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1983:121939 HCAPLUS
DOCUMENT NUMBER: 98:121939
TITLE: H4-isozyme of lactate dehydrogenase in the solution of sodium chloride. 4. Inhibition by oxalate and oxamate
AUTHOR(S): Yamamoto, Sadaaki
CORPORATE SOURCE: Radioisot. Cent., Nagoya Univ., Nagoya, Japan
SOURCE: International Journal of Biochemistry (1983), 15(3), 355-60
CODEN: IJBOBV; ISSN: 0020-711X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The inhibition of H4-isoenzyme of lactate dehydrogenase (H4-LDH) by oxalate and oxamate was studied in 0.5M NaCl. At 20.degree., oxalate inhibition was a mixed type and at 40.degree., the inhibition was uncompetitive. Oxamate inhibition was noncompetitive at low pyruvate concns. and competitive at high pyruvate concns. The inhibition type did not change with temp. An inhibition mechanism is proposed on the basis of a quaternary enzyme complex contg. pyruvate. The distribution of ternary and quaternary enzyme complexes may det. the inhibition type.

=> d ibib abs 15-19

L5 ANSWER 15 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:640363 HCAPLUS
DOCUMENT NUMBER: 129:258972
TITLE: Identification of tumor-associated alleles of genes essential for cell viability and growth and the development of neoplasm inhibitors targeted against them
INVENTOR(S): Housman, David; Ledley, Fred D.; Stanton, Vincent P., Jr.
PATENT ASSIGNEE(S): Variagenics, Inc., USA
SOURCE: PCT Int. Appl., 605 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9841648	A2	19980924	WO 1998-US5419	19980319
WO 9841648	A3	19990429		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9867643	A1	19981012	AU 1998-67643	19980319
EP 973935	A2	20000126	EP 1998-912974	19980319
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1997-41057P P 19970320
WO 1998-US5419 W 19980319

AB Strategies for the identification and targeting of specific alleles of genes in the treatment of tumors are described. Tumor-assocd. alleles of genes coding for proteins essential for cell viability or cell growth and that show loss of an alleles in cancer cells due to loss of heterozygosity (LOH) are identified. Inhibitors of the remaining allele, such as antisense nucleic acids or ribozymes, can then be developed. The method can also be used to inhibit the expression of particular alleles of genes for antigens in the control of transplant rejection. Particular categories of appropriate target genes are described, along with specific exemplary genes within those categories and methods of using such target genes. Antisense phosphorothioate oligonucleotides targeting RNA polymerase II and glutamyl/prolyl tRNA synthetase genes were tested for cytotoxicity in vitro. Oligonucleotides with a single base mismatch were significantly less toxic than those without mismatches. A no. of genes essential for proliferation were mapped and shown to be affected by loss-of-heterozygosity in oncogenesis.

L5 ANSWER 16 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:277932 HCAPLUS
DOCUMENT NUMBER: 130:348640
TITLE: Inhibition of proteolysis of histones in nuclei by nucleotides
AUTHOR(S): Tulenev, V. L.; Konoplich, L. A.; Rudenko, O. A.; Khrapunov, S. N.
CORPORATE SOURCE: Ukraine
SOURCE: Ukrainskii Biokhimicheskii Zhurnal (1998), 70(6), 43-47
CODEN: UBZHD4; ISSN: 0201-8470
PUBLISHER: Institut Biokhimii im. A. V. Palladina NAN Ukrainy
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB Effects of nucleotides on the proteolysis of histones in nuclei incubated at 37.degree. during 1, 3 and 20 h were studied. It has been shown that the H1 histone is removed first during proteolysis and then the H3 and H2B histones are digested. The H4 histone is not cleaved even after 20 h incubation. PMSF and ATP inhibit the H1 cleavage when its structure was not disturbed before. ATP, CTP, ADP, NAD+, AMP and NADH inhibit the partial cleavage of the core histones H3 and H2B. ATP, CTP, AMP and NADH prevent the total digestion of H2B. ATP and, at lower extent, CTP prevent the H3 digestion. ATP, CTP, ADP and NAD+ inhibit the cleavage of the H2A histone in the expts. with 20 h incubation, while H4 is resistant to proteolysis both in the absence and in the presence of nucleotides. The data obtained suggest an important role of ATP and other nucleotides in maintaining the structure of histones and chromatin.

L5 ANSWER 17 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:13848 HCAPLUS
DOCUMENT NUMBER: 128:70747
TITLE: Use of histone deacetylase inhibitors to activate transgene expression
INVENTOR(S): Townes, Tim M.; Chen, Wen Yong; Bailey, Evans C.
PATENT ASSIGNEE(S): UAB Research Foundation, USA
SOURCE: PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 9747307 A1 19971218 WO 1997-US10262 19970613
W: AU, CA, JP
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
AU 9733910 A1 19980107 AU 1997-33910 19970613
PRIORITY APPLN. INFO.: US 1996-664422 19960614
WO 1997-US10262 19970613

AB The invention provides methods for activating transgene expression by administering histone deacetylase inhibitors, methods for identifying compds. that activate transgene expression, and cells that can be used in these screening methods.

L5 ANSWER 18 OF 31 USPATFULL
ACCESSION NUMBER: 94:18720 USPATFULL
TITLE: Method and apparatus for translating differently-sized virtual tributaries organized according to a synchronous optical network (SONET) standard
INVENTOR(S): Afify, Manal, Raleigh, NC, United States
Moore, Allen W., Cary, NC, United States
Hurlocker, Claude M., Raleigh, NC, United States
PATENT ASSIGNEE(S): Alcatel Network Systems, Inc., Richardson, TX, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5291485		19940301	
APPLICATION INFO.:	US 1992-837472		19920218	(7)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Olms, Douglas W.
ASSISTANT EXAMINER: Hom, Shick
LEGAL REPRESENTATIVE: Ware, Fressola, Van Der Sluys & Adolphson
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 2
NUMBER OF DRAWINGS: 36 Drawing Figure(s); 27 Drawing Page(s)
LINE COUNT: 958

AB A SONET virtual tributary reformatter has a drop encoder that receives incoming data of a second VT size mapped according to first VT size and provides the data in a format designed for the first VT size and also has an add decoder that receives outgoing data signals of the second VT size in the format of the first VT size and provides the outgoing data of the second VT size mapped according to the first VT size.

L5 ANSWER 19 OF 31 USPATFULL
ACCESSION NUMBER: 92:77240 USPATFULL
TITLE: Motion vector processing device
INVENTOR(S): de Haan, Gerard, Eindhoven, Netherlands
Huijgen, Hendrik, Eindhoven, Netherlands
PATENT ASSIGNEE(S): U.S. Philips Corporation, New York, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5148269		19920915	
APPLICATION INFO.:	US 1991-727745		19910710	(7)

	NUMBER	DATE
PRIORITY INFORMATION:	EP 1990-201976	19900720

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted

PRIMARY EXAMINER: Kostak, Victor R.
LEGAL REPRESENTATIVE: Goodman, Edward W.
NUMBER OF CLAIMS: 7
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 258
AB In motion compensated interpolation of TV-pictures it is usual to divide the picture into a plurality of blocks, a motion vector being determined for each block. This division into blocks has the disadvantage, that sometimes block boundaries become visible (dirty window effect). The invention generates a continuous vector field from the blocked vector field with the aid of a median filter which calculates for each subblock (H1) from a number of subblocks (H1 . . . H4) into which each block (H) is divided, a motion vector based on the motion vectors of the original block (H) to which the subblock belongs, and of the original blocks (E, G) adjacent to the subblock (H1) concerned.

=> d ibib abs 10-14

L5 ANSWER 10 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:205199 HCAPLUS
DOCUMENT NUMBER: 134:352175
TITLE: Inhibition of histone deacetylation induces constitutive derepression of the beta interferon promoter and confers antiviral activity
AUTHOR(S): Shestakova, Elena; Bandu, Marie-Therese; Doly, Janine; Bonnefoy, Eliette
CORPORATE SOURCE: Laboratoire de Regulation de la Transcription et Maladies Genetiques, CNRS, UPR2228, UFR Biomedicale, Universite Rene Descartes, Paris, 75270, Fr.
SOURCE: Journal of Virology (2001), 75(7), 3444-3452
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The induction of alpha/beta interferon (IFN-.alpha./.beta.) genes constitutes one of the first responses of the cell to virus infection. The IFN-.beta. gene is constitutively repressed in uninfected cells and is transiently activated after virus infection. In this work we demonstrate that histone deacetylation regulates the silent state of the murine IFN-.beta. gene. Using chromatin immunopptn. (ChIP) assays, we show a direct in vivo correlation between the transcriptionally silent state and a state of hypoacetylation of histone H4 on the IFN-.beta. promoter region. Trichostatin A (TSA), a specific inhibitor of histone deacetylases, induced strong, constitutive derepression of the murine IFN-.beta. promoter stably integrated into a chromatin context, as well as the hyperacetylation of histone H4, without requiring de novo protein synthesis. We also show in this work that TSA treatment strongly enhances the endogenous IFN level and confers an antiviral state to murine fibroblastic L929 cells. Inhibition of histone deacetylation with TSA protected the cells against the lost of viability induced by vesicular stomatitis virus (VSV) and inhibited VSV multiplication. Using antibodies neutralizing IFN-.alpha./.beta., we show that the antiviral state induced by TSA is due to TSA-induced IFN prodn. The demonstration of the predominant role of histone deacetylation during the regulation of the constitutive repressed state of the IFN-.beta. promoter constitutes an interesting advance on the understanding of the neg. regulation of this gene and opens up the possibility of new therapeutic perspectives.
REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:887943 HCAPLUS
DOCUMENT NUMBER: 136:353920
TITLE: Inhibition of histone-mediated gene transfer in eucaryotic cells by anti-histone IgG
AUTHOR(S): Hasselmayer, Oliver; Demirhan, Ilhan; Chandra, Angelika; Bayer, Monika; Muller, Roswita; Chandra, Prakash
CORPORATE SOURCE: Gustav-Embden Center of Biological Chemistry, Department of Molecular Biology, Frankfurt University Medical School, Frankfurt, 60590, Germany
SOURCE: Anticancer Research (2001), 21(4A), 2377-2386
CODEN: ANTRD4; ISSN: 0250-7005
PUBLISHER: International Institute of Anticancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In our lab., the gene transfer efficiency of some lipofection reagents (lipofectine, lipofectamine, DOTAP and Dosper) and histones H3 and H4 was compared to that of DEAE-Dextran. The histones H3 and H4 were found to have the highest transfection efficiency of all the agents tested. In the present study we have analyzed other parameters important for gene delivery by the histones H3 and H4. We transferred the HIV-1 tat gene to Jurkat cells and measured the transactivation of HIV-1-LTR by the transactivator protein, expressed in Jurkat cells. The expression of CAT as a reporter gene hybridized to LTR was a direct measure of transactivation potential. In order to investigate whether the transfection was only due to the pos. ionic character of the histones H3 and H4 we tested other histones (H1 and H2A) and polylysine in our system. Under our exptl. conditions, neither polylysine, nor the histones H1 and H2A were able to promote gene transfer in Jurkat cells. The inability of these reagents to promote gene transfer was not dependent on DNA condensation; in EMSA (Electrophoretic Mobility Shift Assay) all these reagents exhibited a strong retardation of DNA. In the presence of anti-histone-IgG the transfection potential of histones H3 and H4 was diminished in a concn. - dependent manner. To investigate whether the histone antibodies inhibited the condensation of DNA by histones we carried out gel retardation assays (EMSA) in the absence and in the presence of histone antibodies. Anti-histone-IgG had no effect on the retardation of histone-DNA complexes; on the contrary, retardation was increased. This observation has led us to postulate two models for the possible mechanism by which the histones H3 and H4 catalyze gene transfer in eucaryotic cells.
REFERENCE COUNT: 82 THERE ARE 82 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 12 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:183622 HCAPLUS
DOCUMENT NUMBER: 135:473
TITLE: Inhibition of histone deacetylase activity by trichostatin A modulates gene expression during mouse embryogenesis without apparent toxicity
AUTHOR(S): Nervi, Clara; Borello, Ugo; Fazi, Francesco; Buffa, Viviana; Pelicci, Pier Giuseppe; Cossu, Giulio
CORPORATE SOURCE: Department of Histology and Medical Embryology, University of Rome, Rome, 00161, Italy
SOURCE: Cancer Research (2001), 61(4), 1247-1249
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal

LANGUAGE: English

AB Remodeling of the chromatin template by inhibition of histone deacetylase (HDAC) activities represents a major goal for transcriptional therapy in neoplastic diseases. Recently, a no. of specific and potent HDAC-inhibitors that modulate in vitro cell growth and differentiation have been developed. In this study we analyzed the effect of trichostatin A (TSA), a specific and potent HDAC-inhibitor, on mouse embryos developing in vivo. When administered i.p. to pregnant mice (at a concn. of 0.5-1 mg/kg) at postimplantation stages (embryonic day 8 to embryonic day 10), TSA was not toxic for the mother and did not cause any obvious malformation during somitogenesis or at later stages of development. Treated embryos were born at similar frequency and were indistinguishable from control animals, developed normally, and were fertile. Interestingly, embryos from TSA-treated mice killed during somitogenesis were modestly but consistently larger than control embryos and presented an increased (+2 to +6) no. of somites. This correlated with an increased acetylation of histone H4, the no. of somites expressing the myogenic factor Myf-5, and the expression of Notch, RAR.alpha.2, and RAR.beta.2 mRNAs. These data indicate that the effects of TSA on transcription: (a) are not toxic for the mother; (b) transiently accelerated growth in mouse embryos without perturbing embryogenesis; and (c) do not result in teratogenesis, at least in rodents. Thus, TSA might represent a nontoxic and effective agent for the transcriptional therapy of neoplasia.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 13 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2001:256736 BIOSIS

DOCUMENT NUMBER: PREV200100256736

TITLE: Cloning of a novel histamine receptor.

AUTHOR(S): Jones, Philip G. (1); Uveges, Albert J. (1); Wu, Shujian (1); Betty, Maria (1); He, Lan (1); Pausch, Mark H. (1)

CORPORATE SOURCE: (1) Wyeth Neurosciences, CN8000, Princeton, NJ, 08543 USA

SOURCE: FASEB Journal, (March 8, 2001) Vol. 15, No. 5, pp. A931. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.

DOCUMENT TYPE: Conference

LANGUAGE: English

SUMMARY LANGUAGE: English

AB The effects of histamine are mediated through 3 G protein-coupled receptors H1-3. Pharmacological evidence suggests that there may be subtypes of H3. Using bioinformatics we have identified the sequence of a novel G protein-coupled receptor encoded on chromosome 18. This clone is 390 amino acids long and BLAST analysis indicates that it's closest relative is the human histamine H3 receptor (43% similarity). The sequence contains the conserved GPCR motifs and of note are the conserved DY residues in transmembrane 3 indicative of muscarinic and histaminergic receptors. Taqman analysis indicates that it is highly expressed in peripheral blood leucocytes. To confirm its identity as a histamine receptor we have expressed the H4 in both yeast and mammalian cells. The coupling of GPCRs to the yeast pheromone pathway provides a universal signaling system for ligand identification, coupling receptor activation to cell growth or reporter gene activity. Using this system we confirm that this receptor is a histamine receptor being stimulated by histamine and R-alpha-methyl histamine. The H3 antagonist thioperamide is also a **H4 antagonist** and interestingly the H3 antagonist clobenpropit acts as a partial agonist. Expression of the receptor in mammalian cells confirms these results and indicates that the H4 is

coupled to the inhibition of adenylyl cyclase.

L5 ANSWER 14 OF 31 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001441392 MEDLINE
DOCUMENT NUMBER: 21378683 PubMed ID: 11387442
TITLE: Methylation of histone H4 at arginine 3 facilitating transcriptional activation by nuclear hormone receptor.
AUTHOR: Wang H; Huang Z Q; Xia L; Feng Q; Erdjument-Bromage H; Strahl B D; Briggs S D; Allis C D; Wong J; Tempst P; Zhang Y
CORPORATE SOURCE: Department of Biochemistry and Biophysics, Lineberger Comprehensive Cancer Center, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599-7295, USA.
CONTRACT NUMBER: GM63067-01 (NIGMS)
P30 CA08748 (NCI)
SOURCE: SCIENCE, (2001 Aug 3) 293 (5531) 853-7.
Journal code: 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010813
Last Updated on STN: 20030105
Entered Medline: 20010823
AB Acetylation of core histone tails plays a fundamental role in transcription regulation. In addition to acetylation, other posttranslational modifications, such as phosphorylation and methylation, occur in core histone tails. Here, we report the purification, molecular identification, and functional characterization of a histone H4-specific methyltransferase PRMT1, a protein arginine methyltransferase. PRMT1 specifically methylates arginine 3 (Arg 3) of H4 in vitro and in vivo. Methylation of Arg 3 by PRMT1 facilitates subsequent acetylation of H4 tails by p300. However, acetylation of **H4 inhibits** its methylation by PRMT1. Most important, a mutation in the S-adenosyl-l-methionine-binding site of PRMT1 substantially crippled its nuclear receptor coactivator activity. Our finding reveals Arg 3 of H4 as a novel methylation site by PRMT1 and indicates that Arg 3 methylation plays an important role in transcriptional regulation.

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L5 ANSWER 6 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:815998 HCAPLUS
DOCUMENT NUMBER: 138:252106
TITLE: Inhibitors of Histone Deacetylation Downregulate the Expression of Endothelial Nitric Oxide Synthase and Compromise Endothelial Cell Function in Vasorelaxation and Angiogenesis
AUTHOR(S): Roessig, Lothar; Li, Huige; Fisslthaler, Beate; Urbich, Carmen; Fleming, Ingrid; Foerstermann, Ulrich; Zeiher, Andreas M.; Dimmeler, Stefanie
CORPORATE SOURCE: Department of Internal Medicine IV, Molecular Cardiology, University of Frankfurt, Germany
SOURCE: Circulation Research (2002), 91(9), 837-844
CODEN: CIRUAL; ISSN: 0009-7330
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The histone deacetylase (HDAC) inhibitor trichostatin A (TSA) inhibits

hypoxia-stimulated angiogenesis. Endothelial nitric oxide synthase (eNOS)-derived NO is central to angiogenesis signaling in endothelial cells (ECs). We hypothesized that the HDAC-dependent regulation of angiogenesis may involve a modulatory effect on eNOS expression. The HDAC inhibitors TSA, butyric acid (BuA), and MS-275 time- and concn.-dependently suppressed eNOS protein levels to 41.+-.2%, 46.+-.12%, and 40.+-.12% of control, resp. In parallel, TSA and BuA also downregulated eNOS mRNA expression to 21.+-.4% and 37.+-.4% of control. TSA also attenuated the NO-dependent relaxation of porcine coronary arteries (P<0.0001, TSA 1 .mu.mol/L) and prevented tube formation in a human angiogenesis assay. Although vascular endothelial growth factor substitution did not compensate for the inhibitory effect of TSA, exogenous NO reversed the inhibition of angiogenesis by TSA. To address the underlying signaling mechanism, we characterized the effect of TSA on eNOS gene transcription and mRNA half-life. Although TSA decreased both eNOS protein and mRNA levels, TSA paradoxically enhanced the activity of the eNOS promoter, and did not alter the eNOS transcription rate in nuclear run-on expts., suggesting that TSA posttranscriptionally targets eNOS mRNA. These data indicate that HDAC-dependent mechanisms contribute to the regulation of eNOS expression in ECs.

REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2003:143696 BIOSIS

DOCUMENT NUMBER: PREV200300143696

TITLE: Pharmacology of Prejunctional Histamine Receptors on Sympathetic Nerves in Isolated Mammalian Irides.

AUTHOR(S): Kulkarni, K. H. (1); LeDay, A. M. (1); Opere, C. A. (1); Ohia, S. E. (1)

CORPORATE SOURCE: (1) Pharmacy Sciences, Creighton University, Omaha, NE, USA USA

SOURCE: ARVO Annual Meeting Abstract Search and Program Planner, (2002) Vol. 2002, pp. Abstract No. 1975. cd-rom. Meeting Info.: Annual Meeting of the Association For Research in Vision and Ophthalmology Fort Lauderdale, Florida, USA May 05-10, 2002

DOCUMENT TYPE: Conference

LANGUAGE: English

AB There is evidence that histamine can inhibit sympathetically-induced papillary dilation in cats by activation of H3-receptors (Koss and Hey, Naunyn-Schmiedeberg's Arch. Pharmacol. 348: 141, 1993). It is, however, unclear whether this action is due to a direct effect of histamine on norepinephrine (NE) release from this tissue. Purpose: The aim of the present study was two-fold: (a) to examine the effect of exogenous histamine on NE release from bovine and human irides and (b) to classify the subtype of histamine receptors mediating this response in bovine irides. Methods: Isolated bovine and human irides were incubated in oxygenated Krebs solution containing 1.6 muM (3H)NE, and flurbiprofen (3 muM) for 1 hour. After incubation, tissues were prepared for studies of (3H)NE release using the superfusion method. Release of (3H)NE was elicited by 300 direct current pulses (supramaximal voltage, 2 ms pulse duration, 5 Hz) applied 84 minutes (S1) and 108 minutes (S2) after the onset of superfusion. Results: Histamine and other receptor selective agonists, R-alpha-methylhistamine (H3-) and imetit (H3-/H4-) caused a concentration-dependent inhibition of field-stimulated (3H)NE release from isolated bovine irides with the following rank order of potency: imetit >> R-alpha-methylhistamine > histamine. All three agonist displayed a similar efficacy in inhibiting evoked (3H)NE release. An equimolar concentration of R-alpha-methylhistamine (1 muM) caused a similar degree of inhibition (35%) of electrically-induced (3H)NE release in both human and bovine

irides. The inhibitory response produced by imetit (1 nM) was completely blocked by thioperamide (30 nM; H3-/**H4-antagonist**). Likewise, the inhibition caused by R-alpha-methylhistamine (1 muM) was abolished by clobenpropit (1 nM; H3-antagonist). Conclusion: We conclude that histamine can inhibit field stimulated (3H)NE release from isolated bovine and human irides. Furthermore, both prejunctional H3- and H4-receptors exist on sympathetic nerve terminals in the bovine irides. These heteroreceptors play an inhibitory role in the regulation of NE release from mammalian irides.

L5 ANSWER 8 OF 31 USPATFULL

ACCESSION NUMBER:	2001:93928 USPATFULL
TITLE:	Logic circuits
INVENTOR(S):	Campbell, Eric R, Hertfordshire, United Kingdom
PATENT ASSIGNEE(S):	Matra BAe Dynamics (UK) Limited, Hertfordshire, United Kingdom (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6249163	B1	20010619
APPLICATION INFO.:	US 1998-80212		19980518 (9)
RELATED APPLN. INFO.:	Continuation of Ser. No. WO 1996-GB2815, filed on 15 Nov 1996		

	NUMBER	DATE
PRIORITY INFORMATION:	GB 1995-23393	19951116

DOCUMENT TYPE:	Utility
FILE SEGMENT:	GRANTED
PRIMARY EXAMINER:	Lam, Tuan T.
LEGAL REPRESENTATIVE:	Nixon & Vanderhye P.C.
NUMBER OF CLAIMS:	7
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	2 Drawing Figure(s); 1 Drawing Page(s)
LINE COUNT:	233

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A transparent latch, having a signal input D, an output Q and a control input C for selecting one of two operating modes, functions by either allowing the output to follow the input (in an enable mode) or by blocking any subsequent changes in input signal level (in an inhibit mode). Owing to the design of the latch, relative propagation delays through gates and interconnecting wires cannot cause a wrong value to be latched, this being in contrast with known arrangements. Therefore constraints on the physical layout of the latch are removed. In one embodiment the latch comprises two pairs of NAND gates (5,6,7,8) each pair being connected in a feedback configuration.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 9 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER:	2001:719165 HCAPLUS
DOCUMENT NUMBER:	136:2144
TITLE:	An Inhibitor-Resistant Histone Deacetylase in the Plant Pathogenic Fungus Cochliobolus carbonum
AUTHOR(S):	Brosch, Gerald; Dangl, Markus; Graessle, Stefan; Loidl, Adele; Trojer, Patrick; Brandtner, Eva-Maria; Mair, Karin; Walton, Jonathan D.; Baidyaroy, Dipnath; Loidl, Peter
CORPORATE SOURCE:	Department of Microbiology, University of Innsbruck Medical School, Innsbruck, A-6020, Austria
SOURCE:	Biochemistry (2001), 40(43), 12855-12863

CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We have partially purified and characterized histone deacetylases of the plant pathogenic fungus Cochliobolus carbonum. Depending on growth conditions, this fungus produces HC-toxin, a specific histone deacetylase inhibitor. Purified enzymes were analyzed by immunoblotting, by immunopptn., and for toxin sensitivity. The results demonstrate the existence of at least two distinct histone deacetylase activities. A high mol. wt. complex (430 000) is sensitive to HC-toxin and trichostatin A and shows immunoreactivity with an antibody against Cochliobolus HDC2, an enzyme homologous to yeast RPD3. The second activity, a 60 000 mol. wt. protein, which is resistant even to high concns. of well-known deacetylase inhibitors, such as HC-toxin and trichostatin A, is not recognized by antibodies against Cochliobolus HDC1 (homologous to yeast HOS2) or HDC2 and represents a different and/or modified histone deacetylase which is enzymically active in its monomeric form. This enzyme activity is not present in the related filamentous fungus Aspergillus nidulans. Furthermore, in vivo treatment of Cochliobolus mycelia with trichostatin A and anal. of HDACs during the transition from non-toxin-producing to toxin-producing stages support an HC-toxin-dependent enzyme activity profile.
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> log h

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	61.93	63.39

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-9.77	-9.77

SESSION WILL BE HELD FOR 60 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 15:29:38 ON 13 JUN 2003